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In situ capture of mu-calpain activation in platelets.

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Department of Molecular Biology, Tokyo Metropolitan Institute of Medical Science, Japan.

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In situ detection of calpain activation in intact cells has not been possible to date. Here we present the first direct evidence, employing a novel approach, that mu-calpain is rapidly activated at cell membranes in platelets upon a rise in intracellular calcium concentration. Immunoelectron microscopy using antibodies capable of distinguishing between the pre- and postautolysis forms of mu-calpain revealed that treatment of platelets with calcium ionophore causes the preautolysis form of the protease to translocate from cytosol to membranes, where it becomes activated by autolysis. This indicates that proteins associated with membranes serve as primary substrates for calpain in cells.

PMID: 8463275 [PubMed - indexed for MEDLINE]

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